

Trial by trial effects in the antisaccade task

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ABSTRACT

The antisaccade task requires participants to inhibit the reflexive tendency to look at a sudden onset target and instead direct their gaze to the opposite hemifield. As such it provides a convenient tool with which to investigate the cognitive and neural systems that support goal-directed behaviour. Recent models of cognitive control suggest that antisaccade performance on a single trial should vary as a function of the outcome (correct antisaccade or erroneous prosaccade) of the previous trial. In addition, repetition priming effects suggest that the spatial location of the target on the previous trial may also influence current trial performance. Thus an analysis of contingency effects in antisaccade performance may provide new insights into the factors that influence the monitoring and modulation of the antisaccade task and other ongoing behaviours. Using a multilevel modelling analysis we explored previous trial effects on current trial performance in a large antisaccade dataset. We found (1) repetition priming effects following correct antisaccades; (2) contrary to models of cognitive control antisaccade error rates were increased on trials following an error, suggesting that failures to adequately maintain the task goal can persist across more than one trial; and (3) current trial latencies varied according to the previous trial outcome (correct antisaccade, slowly corrected error or rapidly corrected error). These results are discussed in terms of current models of antisaccade performance and cognitive control and further demonstrate the utility of multilevel modelling for providing a powerful statistical technique for analysing antisaccade data.

Keywords: Saccades, Attention, Intention, Memory-Short Term, Inhibition(Psychology)

INTRODUCTION

Complex behaviour requires the monitoring of ongoing action and subsequent behavioural adjustment in order to prevent, detect and (if necessary) correct erroneous responses (Dehaene et al., 2003; Dehaene & Naccache, 2001; Dehaene, Kergsberg & Changeux, 1998; Mayr, 2004; Mayr, Awh & Laurey, 2003; Miller & Cohen, 2001). This monitoring – or cognitive control - is particularly important when execution of the correct behavioural response requires the suppression of an over-learned or otherwise pre-potent response. In circumstances such as these, “action slips” may occur, particularly if we are distracted or otherwise fail to adequately maintain the original goal or intention (e.g., James 1890; Norman, 1981; Reason, 1984). A convenient laboratory analogue of action slips is provided by the antisaccade task (Hallett, 1978). Observers are required to suppress a highly pre-potent prosaccade towards a sudden onset target and instead make an eye movement in the opposite direction, often to an equidistant position in the opposite hemifield. Healthy participants typically fail to suppress erroneous prosaccades towards the target on about 20-25% of trials, before correctly saccading towards the mirror image location (e.g., Fischer & Weber, 1992; Smyrnis et al, 2002).

Comparatively few studies have systematically explored the processes involved in monitoring and adjusting performance during saccadic eye movement tasks on a trial by trial basis. Those that do have focussed on repetition priming effects – the extent to which repeating the stimulus location and/or saccade direction from one trial to the next impacts on performance (Fecteau, Au, Armstrong & Munoz, 2004; Nieuwenhuis, Ridderinkhof, Blom, Band & Kok, 2001; Barton, Goff & Manoach, 2006). Current models of cognitive control suggest that our response to the *outcome* of the previous trial (e.g., correct or error) may also impact on current trial performance (Botvinick et al, 2001). An example of such behaviour from reaction time tasks is “post-error slowing” - a shift in the trade off between speed and

accuracy to a more cautious response mode which occurs on the trial following an erroneous response (Botvinick et al, 2001; Hodgson et al, 2004; Rabbitt, 1966; Rabbitt & Rodgers, 1977). A critical distinction between two currently influential accounts of cognitive control concerns the role of conscious awareness. According to the conflict monitoring model outlined by Miller & Cohen (2001) the dorsolateral prefrontal cortex (DLPFC) and anterior cingulate cortex (ACC) comprise a closed feedback circuit, with the ACC signalling for increased cognitive control from the DLPFC whenever conflict is detected (either consciously or unconsciously). In contrast, according to Dehaene et al's account (Dehaene et al., 2003; Dehaene & Naccache, 2001; Dehaene et al., 1998), neural and behavioural indications of increased cognitive control should be observed only when *conscious* awareness of conflict is high (Mayr, 2004).

The antisaccade task provides an excellent opportunity to test specific predictions arising from these two alternative accounts of cognitive control, as it has recently been demonstrated that participants are unaware of around 50% of the errors they make (Mokler & Fischer, 1999; Nieuwenhuis et al., 2001). Both recognised and unrecognised errors are usually corrected by one or more saccades in the opposite direction to the initial prosaccade, but recognised (conscious) errors are typically corrected more slowly than unrecognised (unconscious) errors. Research using other paradigms has suggested that errors that are corrected rapidly are dissociable from those that are corrected more slowly both in terms of conscious awareness and electrophysiological responses (Rodriguez-Fornells, Kurzbuch & Münte, 2002; Fiehler, Ullsperger & Cramon, 2005). These previous studies have, however, only considered these differences within the timecourse of a single trial.

In addition to changes in control afforded by conflict monitoring or error detection systems, other factors have been shown to influence antisaccade performance on a trial by trial basis. In particular, given that the antisaccade task employs stimuli that are typically distinguished only by hemisphere of presentation and occasionally by eccentricity, certain

“repetition priming” effects, (whereby the processing of a given stimulus is facilitated if it occurred on a previous trial) may be expected (Mayr et al., 2003; Soetens, 1998; Fecteau & Munoz, 2003; Fecteau, et al., 2004; Barton et al., 2006). For example, in a simple prosaccade task, Dorris, Paré and Munoz (2000) demonstrated that saccadic latencies in non-human primates are reduced if the target occurs in the same spatial location as on a previous trial, and increased if it occurs in the diametrically opposite position. In humans, the opposite pattern has been observed; latencies are increased if the stimulus is repeated, but reduced if the hemifield of presentation alternates between trials (Fecteau et al, 2004; Barton et al., 2006).

The research outlined above suggests that a variety of factors may influence ongoing performance during the antisaccade task. Models of cognitive control suggest that increased control following erroneous trials may result in post-error slowing, and an increased likelihood of a correct response. Repetition priming effects suggest that the spatial location of the target on the preceding trial may also impact on current trial performance. Current models of antisaccade performance (e.g., Munoz & Everling, 2004) allow for both of these effects to be mediated via changes in the baseline activity in the neural systems mediating saccadic responses, but only repetition priming effects have been investigated. The purpose of the present study was to determine the effects of, and interactions between these factors on antisaccade performance in a large sample of trials. An increased understanding of the role of these processes would provide further insights into why antisaccade errors are increased in certain patient populations, and also inform current models of cognitive control.

METHOD

Participants. Ninety undergraduates from the University of Sussex contributed data for this study. All had normal or corrected to normal vision. Participants were naïve to the purposes of this study. All participants provided written consent. The study was carried out in accord with the principles laid down in the Helsinki Declaration and was approved by the School of Life Sciences Ethics Committee.

Apparatus. Stimuli were displayed using a Viewsonic P225f 22" pure flat CRT monitor at a resolution of 1,280 x 1,024 pixels, with a refresh rate of 85 Hz. Participants viewed the stimuli from a distance of approximately 60 cm. Eye movements were recorded using an SR Research Ltd., EyeLink II eye tracker, with a temporal resolution of 2 ms and a spatial resolution better than 0.25 degrees. A 3-point horizontal target array was used for eye tracker calibration. A second 3-point array was used to validate this calibration.

The antisaccade task. Prior to each experiment, the participants were instructed that when the peripheral target appeared they should not look at it but should instead move their eyes as quickly and as accurately as possible to the mirror-image location (an equal distance from central fixation but in the opposite direction). Each trial began with the presentation of a central fixation marker (a filled red circle measuring 0.5 degrees of visual angle in diameter, luminance 15 cd/m), on a black background (average luminance 4.2 cd/m). After a brief period the central marker was extinguished and there followed a 200 ms period in which the screen was blank (the gap period). The period before the central marker was extinguished allowed a drift-correct procedure to be performed and varied between 600 ms and 3,100 ms. After the 200 ms gap, a peripheral target appeared (another filled red circle measuring 0.5 degrees in diameter) in one of four locations: -8 degrees, -4 degrees, +4 degrees and +8

degrees from the position at which the central fixation marker had been displayed. The target remained on screen for 2000 ms. The location at which the stimulus appeared was varied randomly, with an equal probability of appearing in any of the four possible locations. Figure 1 shows a schematic of an antisaccade trial. Participants each contributed data from between two and eight blocks of 72 antisaccade trials.

FIGURE 1 ABOUT HERE

Data analysis. Analysis was carried out off-line after completion of the experiment. Initial extraction of the eye position data was carried out using software supplied with the EyeLink II eye tracking system: Saccade detection required a deflection of greater than 0.1° , with a minimum velocity of 30°s^{-1} and a minimum acceleration of 8000°s^{-2} , maintained for at least 4 ms. No minimum fixation duration criterion was imposed. Further analysis was conducted using custom software developed within the MATLAB programming environment. Trials were excluded if the centre of gaze was not within 50 pixels (approximately 1.25 degrees) of the central fixation marker when the peripheral target appeared as an eccentric position of gaze when the target appeared would confound our study of the influence of target location. Trials were also excluded if saccades were initiated within 80 ms of the appearance of the peripheral target as these are unlikely to have been generated in response to the target, even given express saccade latencies (e.g., Fischer and Weber, 1993; Wenban-Smith and Findlay, 1991); such rapidly initiated saccades are likely to be anticipatory. Trials were also excluded if no eye movements were initiated within two seconds of the appearance of the peripheral target. As a result of these selection criteria, a total 21,901 trials were available for subsequent analysis.

Statistical analysis. In traditional analyses of antisaccade data, the relevant performance indices (e.g., error rate, average correct antisaccade latency, average primary saccade amplitude, etc.) are calculated individually for each participant, and these values submitted to analysis of variance (ANOVA). By aggregating across trials and treating the participant as the unit of analysis, potentially interesting trial level information is lost. Even if trial level data are not the focus of the analysis, it is worth noting that this standard ANOVA approach treats means based on a single trial or small number of trials (for example for correct antisaccade latency if the participant only makes one or two correct responses) as precise, and given equal weight to a mean based on a substantial number of trials. Techniques based upon analyses of variance therefore become particularly problematic for data in which the number of samples contributed by individuals varies widely, as in the present study.

In this paper we were primarily concerned with how the performance of the antisaccade task on the current trial is influenced by the previous trial. This type of analysis requires that each trial is treated as an individual data point, rather than using participant means. Treating individual trials as data points in a traditional ANOVA violates the assumption of independence, as some participants make more errors than others, and average saccade latency varies across subjects. Treating trials as a repeated measures factor takes such dependencies into account, but leads to excessively complex models (when the number of trials is large), and the model must be balanced in that all participants must contribute the same number of repeated observations. An appropriate technique for an analysis of this type is to use multilevel models, also known as hierarchical linear models or random coefficient models (Bryk & Raudenbush, 1992; Goldstein, 1995). This approach recognises the hierarchical nature of many datasets, and is ideal for analysing data from tasks such as the antisaccade, where trials are nested within participants. In particular this technique is robust to missing data points, and does not require similar numbers of observations in each cell of the experimental design. It should be noted that our multilevel modelling approach does not

forfeit ANOVA's advantage of being able to generalise across participants: Participants are treated as a random variable, and as such the analysis allows generalisation across the participant population, and to participants in general.

The basic multilevel model is similar to the standard linear model

$$y_{ij} = \beta_0 + \beta_1 x_{ij} + e_{ij} \quad (1)$$

where dependent variable y is a function of predictor variable x . The subscripts indicate a repeated measures type model where the responses of several individuals (j) are measured on several different occasions (i). β_0 is the intercept and β_1 the regression coefficient. The error associated with each observation in terms of the difference between its actual value and that predicted by the regression coefficients is represented by e_{ij} (the residuals). In conventional regression models the coefficients are fixed and have the same value for each participant. In multilevel, or hierarchical models, the regression coefficients are free to vary between participants. In the multilevel approach the model is modified to allow different participants to have different slopes and intercepts (β):

$$y_{ij} = \beta_0 + u_{0j} + \beta_1 x_{ij} + u_{1j} x_{ij} + e_{ij} \quad (2)$$

In this equation u_{0j} and u_{1j} are random quantities and represent participant j 's deviation from the average β_0 and β_1 respectively. For repeated measures data a simple random intercept model is sufficient:

$$y_{ij} = \beta_0 + u_{0j} + \beta_1 x_{ij} + e_{ij} \quad (3)$$

The first level of the analysis (i) represents the trial, and the second level (j) the participant. The intercept term varies randomly across participants and the effects of the explanatory variables are assumed to be constant across participants. In multilevel models explanatory variables can be measured at each level. In the present analysis all explanatory variables are at the trial level, but the approach can also accommodate participant level variables, and their

interaction with trial level variables, making it suitable for research comparing antisaccade performance in different populations (such as patients versus controls). A further advantage of ML models, particularly relevant for antisaccade data, is that they can easily accommodate binomial data (such as trial outcome: correct vs error). For binomial data the model uses a multilevel logistic regression.

All analyses were conducted using MLwiN (version 2.0, Multilevels Model Project, Institute of Education, University of Bristol). Significance testing was performed with log-likelihood ratio tests that compare nested models with reduced models that do not include the variables of interest. The likelihood ratio test follows a chi-square distribution, with q degrees of freedom, where q is the difference in the number of parameters between the two models.

Initially we considered general antisaccade performance characteristics for our data set. We measured error rate, primary saccade latency, and the latency with which prosaccade errors were corrected (as measured from the offset of the erroneous prosaccade). All primary saccades that met the criteria for saccade detection were included in the analysis. For our analysis of contingency effects in the antisaccade task, we created a number of binary dummy variables to code the variables of interest. In order to establish whether any apparent effects of the previous trial resulted from longer term trends such as learning or fatigue we coded trials within each block as either first half or second half. There were no significant effects of this variable on either error rate or latency so for clarity and simplicity is not included in the analyses presented below. The other dummy variables were current trial outcome (coded as either correct or error), previous trial outcome (also coded as correct or error) and target hemifield congruency: i.e. whether the target on the current trial appears in the same hemifield as on the previous trial (coded as congruent or incongruent).

RESULTS

General performance metrics

The average error rate was 24.1% (SD = 8.2). This is comparable with previous studies (e.g., Evdokimidis et al., 2002; Everling & Fischer, 1998; Mokler & Fischer, 1999). Participants made more errors when the target appeared to the right of fixation (26.2% of trials) than when it appeared on the left (22.1%; $\chi^2(1) = 49.173$, $p < 0.01$). This finding is consistent with the majority of recent reports (e.g., Bell, Everling & Munoz, 2000; Smyrnis et al., 2002).

As expected, erroneous prosaccades had shorter latencies ($M = 139.0$, $SD = 28.6$ ms) than correct antisaccades ($M = 239.0$ ms, $SD = 38.1$ ms, ; $\chi^2(1) = 11842$, $p < 0.01$). The increase in latencies observed for correct antisaccades is generally assumed to reflect the additional processing involved in inhibiting the reflexive prosaccade and performing the spatial transform required to provide the co-ordinates for the antisaccade, although the relative importance of these processes remains unclear (Evdokimidis et al., 2002; Olk & Kingstone, 2002, 2003). When compared to the baseline model, target hemifield had no impact on primary saccade latency. However, compared to a model containing the effects of current trial and target hemifield, a model containing their interaction led to a significant improvement in model fit ($\chi^2(1) = 41.0$, $p < 0.01$): Correct antisaccades were made slightly more quickly if the target appeared in the left hemifield (236.3 ms) than if it appeared on the right (241.9 ms). Conversely, antisaccade errors had a shorter latency if the target appeared in the right hemifield (136.5 ms) than if it appeared on the left (141.9 ms). An antisaccade away from a target in the left hemifield is a rightward eye movement. Similarly a prosaccade toward a target in the right hemifield is a rightward movement. Thus in both cases latencies were shorter when the eye movement that was initiated was rightward. Although the interaction is

significant, it is worth noting that the differences are very small (around 5 ms or less) and are perhaps unlikely to be of particular behavioural significance.

When erroneous prosaccades were generated, they were corrected by a saccade in the opposite direction before the end of the trial in 99.5% of cases. The mean latency before these errors were corrected was 131.4 ms (SD = 54.8) consistent with previous research (Evdokimidis et al., 2002; Massen, 2004; Mokler & Fischer, 1999). Like these previous studies, we found that a considerable proportion of these correction latencies were very short: 60.3% of the errors were corrected within 120 ms and 27.3% were corrected within 80 ms.

Trial-by-trial contingencies

Recent evidence suggests that errors can have differential effects both within and beyond the time course of a trial. Mokler and Fischer (1999) found that errors that the participant is aware of making tend to have longer correction latencies than those that they are unaware of. Dehaene and colleagues (Dehaene et al., 2003; Dehaene & Naccache, 2001; Dehaene et al., 1998) argue that increased cognitive control should only occur after errors of which the participant is aware. In order to explore the possibility of differential inter-trial effects for errors in the antisaccade task (and in the absence of subjective feedback from participants), we split our errors into two groups based on the mean latencies for recognised and unrecognised errors described by Mokler & Fischer, 1999 – rapidly corrected (<80 ms) or slowly corrected (>150 ms).

Error rates

Our analysis of error rates compared the effects of, and the interactions between previous trial outcome and hemifield congruence. Previous trial outcome specified whether

the primary response on the previous trial was a correct antisaccade, a rapidly corrected erroneous prosaccade or a slowly corrected erroneous prosaccade. The incidence of errors varied according to the outcome of the previous trial ($\chi^2(2) = 18.0$, $p < 0.01$). Antisaccade errors were lower if the previous trial was a correct antisaccade (22.9%), compared to an error that was corrected within 80 ms (26.4%; $\chi^2(1) = 7.69$, $p < 0.01$, $N = 1353$ errors) or an error that was corrected with a latency in excess of 150 ms (27.6%; $\chi^2(1) = 11.72$, $p < 0.01$, $N = 1066$ errors). The difference in error rates following slowly- and quickly-corrected errors was not significant $\chi^2(1) = 0.4$. We accept that our boundaries of 80 ms and 150 ms are essentially arbitrary. The analyses were repeated with several alternative boundaries, both wider and narrower, and the critical findings remained significant.

Error rates also varied with hemifield congruence ($\chi^2(1) = 73.8$, $p < 0.01$) with errors occurring less often if the target appeared in the same hemifield as on previous trials (21.0%) compared to trials on which it appeared in a different hemifield on the previous trial (26.2%). The interaction between hemifield congruence and previous trial outcome was also significant $\chi^2(2) = 6.02$, $p < 0.05$ (Table 1). On trials following a correct antisaccade, error rates were lower if the target appeared in the same hemifield as it had on the previous trial (19.9%) than if it appeared in the opposite hemifield (25.7%). There were no effects of hemifield congruence on trials following a rapidly corrected error or on trials following a slowly corrected error.

TABLE 1 ABOUT HERE

Primary saccade latency

A model containing the parameter coding for current trial outcome (correct vs. error) was used as the baseline model. Models containing previous trial outcome ($\chi^2(2) = 31.7$, $p < 0.01$), and the interaction between previous and current trial outcome ($\chi^2(2) = 79.6$, $p < 0.01$)

both led to a significant increase in model fit. As can be seen in Figure 2, the interaction occurs because the previous trial outcome has opposite effects on the latency of correct antisaccades and erroneous prosaccades. For correct antisaccades on the current trial (Figure 2, left), the latency was 239.6 ms if the previous trial outcome was also a correct antisaccade. However, for correct antisaccades on trials following a rapidly corrected error on the previous trial, this latency reduced to 224.6 ms. Conversely, for correct antisaccades on trials following slowly corrected errors on the previous trial, latency increased to 248.2 ms. When the current trial outcome was an erroneous prosaccade (Figure 2, right), the latency of this response was 139.2 ms if the previous trial outcome was a correct antisaccade. For error prosaccades on trials following a rapidly corrected error on the previous trial, this latency increased to 145.7 ms. for error prosaccades on trials following a slowly corrected error on the previous trial the latency of the erroneous prosaccade was reduced to 130.0 ms.

FIGURE 2 ABOUT HERE

The only other model that led to a significant reduction in the log likelihood ratio compared to the baseline model of current trial outcome, was a model that included the interaction between hemifield congruency and current trial outcome. The interaction occurs because correct antisaccades are slightly slower if the target appears in the opposite hemifield to the previous trial (240.3 ms) than if it appears in the same hemifield (237.8 ms); whereas, antisaccade errors are slightly faster if the target appears in the opposite hemifield to the previous trial (137.1 ms) than if it appears in the same hemifield (142.3 ms). Hemifield congruency did not interact with previous trial outcome.

DISCUSSION

A contingency analysis of a large sample of antisaccade trials revealed three key findings. First, repetition priming effects were observed following correct antisaccades. Second, we found that antisaccade error rates were increased on trials following an error. Third, current trial latencies varied according to the previous trial outcome (correct antisaccade, slowly corrected error or rapidly corrected error).

Repetition priming in the antisaccade task

Following a correct antisaccade, if the target appeared in the same hemifield on the next trial, the error rate was low and the primary saccade latency for correct antisaccades was also slightly reduced. This result suggests that when an antisaccade program is executed, activation within that pathway may remain elevated for the subsequent trial. Thus there is facilitation of that pathway (an antisaccade in a particular direction) in the following trial. Such a priming effect is consistent with previous reports of repetition priming (Dorris et al., 2000; Mayr et al., 2003; Soetens, 1998). That this particular repetition priming effect is confined to trials following correct antisaccades is not unexpected: On error trials both the incorrect program (the prosaccade) and the correct antisaccade are executed, thus making a selective priming of one of these pathways in the next trial unlikely. Barton, Goff and Manaoch (2006) found repetition priming effects on pro and antisaccade latencies, but these were related to the penultimate trial (N-2), not the previous trial (N-1).

A recent account of antisaccade performance assumes that a “competition” ensues at stimulus onset between the exogenously triggered prosaccade and the endogenously initiated antisaccade (e.g., Munoz & Everling, 2004; Massen, 2004); the pathway that reaches

threshold first is executed (see Mokler & Fischer, 1999; Schlag-Rey, Amador, Sanchez & Schlag, 1997; Trappenberg, Dorris, Munoz & Klein, 2001). Reaching threshold for either pathway can be modified either by changing the rate at which activation of the pathway rises to threshold, or the baseline activation in the pathway prior to stimulus onset (see Carpenter, 1981; Carpenter & Williams, 1995; Hanes & Carpenter, 1999). Variations in baseline activation have been suggested for simple reflexive saccadic tasks: inter-trial effects have been interpreted as reflecting residual activation in topographic *saliency maps* that persist beyond the duration of a single trial (Fecteau and Munoz, 2003). Dorris and colleagues (Dorris & Munoz, 1995; Dorris, Taylor, Klein & Munoz, 1999; Dorris et al., 2000) suggested that inter-trial variations in baseline activation is not limited to a topographic saliency map: location-independent “pre-target factors” can also result in variations in the baseline activity for particular saccadic programs, upon which post-target activity accumulates. It is possible that our observed repetition priming effect reflects increased activity in the antisaccade pathway for one particular hemifield, facilitating correct antisaccade if the target appears in the same hemifield on the next trial.

Between each trial, one or more saccades were made to return the centre of gaze to the central pre-trial marker. Given that most errors are followed by an antisaccade to the mirror location, the final re-centring saccade almost always takes the form of a saccade from the target’s mirror location to the centre of the display. It may be that some of the observed priming effects arise from this return saccade rather than the initial response by the observer. Since the occurrence of this return saccade was not manipulated, it is not possible to untangle the potential influence of this from the influence of the initial response. However, because we find differential inter-trial effects depending upon the outcome of the previous trial, yet the return saccade is relatively constant across different previous trial outcomes, we feel that the reported results are unlikely to arise solely from the return saccade.

Increased error rates after errors

Executing an erroneous prosaccade on the previous trial resulted in an increased probability of making another error on the current trial. There are two possible explanations for this result: priming of the error response pathway, or prolonged goal neglect.

Executing an erroneous prosaccade rather than an antisaccade suggests that the prosaccade pathway received more activation than the antisaccade pathway on the previous trial. It would appear that this pathway may remain primed for the next trial and therefore increase the probability of the erroneous prosaccade reaching threshold before the correct response. Interestingly, this priming effect in the erroneous pathway is not location- (or direction-) specific, but is a general priming of the erroneous stimulus-driven response.

An alternative explanation to our suggestion of error-response-priming is that successive errors result from a transient failure to adequately maintain the goal of the task. That is, rather than the previous error resulting in elevated baseline activation in the prosaccade pathway on the current trial, goal neglect could result in lowered baseline activation in the antisaccade pathway (see also Nieuwenhuis, Broerse, Nielen and de Jong, 2004). Whether arising from pathway-specific priming or goal neglect, we find that this effect frequently persisted beyond the timescale of a single trial: sequences of consecutive errors were common, and, critically, the probability of making an error on any given trial increased dramatically with the number of erroneous preceding trials (Table 2).

TABLE 2 ABOUT HERE

It should be noted, that our observation of an increased probability of making an error following an error on the previous trial is opposite to the predictions made by the popular conflict monitoring account of antisaccade performance (e.g., Botvinick et al., 2001; Laming, 1968; Miller and Cohen, 2001).

Differential effects of slowly- and rapidly-corrected errors on the following primary saccade latency

The conflict monitoring account of antisaccade performance predicts that an error would lead to slower response on the following trial (e.g., Botvinick et al., 2001; Laming, 1968; Miller and Cohen, 2001). Indeed, in situations where conflict and cognitive control are high, slower responses are often found after an error (Rabbitt, 1966; Rabbitt & Rodgers, 1977; Hodgson et al., 2004). Our data show that post-error slowing can be found in certain circumstances, but not in all situations. Post-error slowing was found for correct antisaccades on trials following an error that was corrected with a latency in excess of 150 ms on the previous trial. However, if the error on the previous trial was corrected rapidly (with a latency of less than 80 ms) no post-error slowing was observed; rather a post-error *quickening* was found. Why this differential influence of rapidly- and slowly-corrected errors might arise is beyond the scope of the present data.

Our observation of an increased correct antisaccade latency only following slowly-corrected errors may be because post-error slowing is restricted to trials following errors of which the participant is aware (Nieuwenhuis et al., 2001) and consciously-recognised errors tend to have longer latencies (Mokler & Fischer, 1999).

The post-error quickening of correct antisaccade responses that we observed after rapidly corrected errors is an interesting and novel finding and cannot easily be accounted for within current accounts of antisaccade performance. One speculation concerns the anterior cingulate cortex (ACC): increased activation in the ACC is typically associated with error detection and conflict monitoring (e.g., Falkenstein, Hohnsbein, Hoormann & Blanke, 1991; Falkenstein, Hoormann, Christ & Hohnsbein, 2000; Gehring, Goss, Coles, Meyer & Donchin, 1993; Nieuwenhuis et al., 2001), but has also recently been implicated in promoting a state of

increased general arousal (Critchley et al., 2003). It may be that increased activity in the ACC arising from error detection increases general arousal and thus elevates activity in both the prosaccade and antisaccade pathways.

While for correct antisaccadic responses on the current trial a post-error slowing was found following slowly corrected errors and a post-error quickening was found following rapidly corrected errors; for erroneous prosaccades on the current trial the opposite pattern was observed: error latencies were increased slightly following rapidly corrected errors, and reduced slightly following slowly corrected errors. Current accounts of antisaccade performance do not predict this result and at present we can find no obvious explanations; it will be important to see whether it is replicated in future studies.

Conclusion

The outcome of a trial can have pronounced influences upon antisaccade performance in the subsequent trial. Post-error slowing, predicted in many conflict monitoring accounts of antisaccade performance, is only observed for correct antisaccades following slowly corrected errors on the previous trial. If an error is corrected rapidly, no such post-error slowing is observed on the next trial; indeed in these situation post-error quickening ensues. Periods of goal neglect during the task are apparent that may span several trials: errors are likely to be followed by errors on the next trial and even for several successive trials. Repetition priming effects also influence antisaccade performance: after correct antisaccades, correct antisaccades into the same hemifield are promoted. Not only do our data allow us to consider the trial-by-trial modulation of the antisaccade task, but they also demonstrate the utility of multilevel modelling techniques for analysing antisaccade data. Using this technique it is possible to look at fine-grained differences between individual trials while also accounting for between-observer differences (both of these aspects are crucial components to account for in

the antisaccade task). Our data clearly demonstrate the need to consider trial-by-trial contingencies in any account of antisaccade performance, and suggest a similar requirement to account for such effects in models of ongoing cognitive control of behaviour. Further research employing detailed analyses of trial-by-trial contingencies - in particular, considering effects across a series of consecutive trials - in antisaccade performance using techniques such as multilevel modelling will help develop our understanding of the cognitive processes underlying the ongoing monitoring and modification of task performance, and might provide important clues as to why and in what ways errors are increased in various clinical populations.

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FIGURE LEGENDS

FIGURE 1. The antisaccade task. The grey inset box shows a schematic of the trial sequence. A central fixation marker was visible for between 0.6 and 3.1 seconds. This was followed by a 200 ms gap period where no target was visible. After this the peripheral target appeared in one of four locations. In this schematic, the target appears 8 degrees to the left of fixation (alternative locations are shown by the dotted rings, but no such rings were visible to the observer). The plot shows example responses from trials on which the target appeared 8 degrees to the left of fixation. Example recordings are shown for a correct antisaccade (black trace) and an erroneous antisaccade (grey trace). Note that the erroneous prosaccade depicted here was followed by a saccade in the opposite direction: to the target's mirror location (after about 100 ms). Our three primary measures of antisaccade performance are also depicted on this plot. For correct antisaccades we measured the latency of the primary (antisaccade) response (L_{AS}). For error prosaccades we measured the latency of the primary response (L_{Er}) and then the latency with which the error was corrected (L_{Corr} : the time between the end of the erroneous prosaccade and the start of the antisaccade).

FIGURE 2. The influence of previous trial outcome and current trial outcome upon primary saccade latency. Correct antisaccades were generally slower than erroneous prosaccades. After a rapidly corrected error (light grey bars), correct antisaccades had shorter latencies than after a correct antisaccade on the previous trial (black bars); showing post-error quickening. After a slowly corrected error (dark grey bars), the latencies of correct antisaccades were longer than after correct antisaccades (black bars); showing post-error slowing. The opposite pattern of influence of slowly- and rapidly-corrected errors on primary saccade latency was observed following an erroneous prosaccade on the previous trial.

	Correct antisaccade on previous trial	Rapidly corrected error prosaccade on previous trial	Slowly corrected error prosaccade on previous trial
Same hemifield as previous trial	19.86%	25.73%	26.19%
Opposite hemifield to previous trial	25.75%	26.91%	28.99%

TABLE 1. The influence of hemifield congruence upon error rates on trials following correct antisaccades, rapidly corrected errors (correction latency < 80 ms), or slowly corrected errors (correction latency > 150 ms).

Error Sequence Length	1	2	3	4	5	6+
Number of Sequences	2158	521	162	81	35	41
Probability of Making an Error	.34	.44	.53	.56	.62	-

TABLE 2. The frequencies with which sequences of consecutive errors were executed, and the probability of making an error after an error sequence of that length. Note that the frequencies refer to the number of discrete sequences of each length (e.g., there were 81 instances where 4 consecutive errors were made before a correct trial) whereas the probabilities refer to the likelihood of an error occurring on the next trial after any error sequence of a given length (e.g. after 2 errors which may occur in a longer sequence of errors).